REMARKS

Claims 11-27 are pending. Claims 13-20 are withdrawn. Claims 11, 12, and 21-27 are rejected.

CLAIM REJECTIONS UNDER 35 U.S.C. §103

Claims 11, 12, and 21-27 are rejected under 35 U.S.C. §103(a) as obvious over Cuttitta in view of Pandurangi.

All claims are now amended to recite that Applicants' method, which is directed to and recites "a phototherapeutic procedure" that causes photoexcitation of the Ar and N_3 components, "results in tissue damage" which thereby treats the target tissues.

The amendment is supported at least in ¶7 of the published application (U.S. 2204/0180864) and thus introduces no new matter. Specifically, ¶¶7-9 describe how type 1 mechanisms operate: the photosensitizer (also abbreviated as "sensitizer"), in the presence of light, directly and via the lowest triplet state transfers electrons or energy to cell components without requiring oxygen to achieve cellular injury, as explained at ¶12 and thus distinguishing from type 2 mechanisms. Moreover, the published application at ¶10 explains that "the biological basis of tissue injury brought about by tumor phototherapeutic agents has been the subject of intensive study", and goes on to describe seven "various biological mechanisms postulated" (denominated (a)-(g)). These mechanisms depend, at least in part, on the specific target tissue. Cancer cells, for example, may upregulate expression of low density lipoprotein (LDL) receptors (mechanism (a)) where the agents selectively bind to LDL and albumin; and/or undergo apopotosis induced by photosensitizers (mechanism (g)). Tumors, for example, often contain increased number of lipid bodies and are thus able to bind to hydrophobic photosensitizers (mechanism (c)); may have increased capabilities for phagocytosis or pinocytosis of porphyrin aggregates (mechanism (e)); and macrophages associated with them may be responsible for the concentration of photosensitizers in tumors (mechanism (f)). Vasculature, for example, may be "leaky" and also have reduced lymphatic drainage, causing porphyrin accumulation (mechanism (d)), and proliferative neovasculature may selectively take up porphyrin-like substances (mechanism (b)).

Cuttitta, cited "for the teaching that receptor specific administration were [*sic*] known in the art, specifically compounds used to bond to bombesin receptors", and "not cited for the phototherapy component" (October 25, 2010 Advisory Action), in view of Pandurangi, does not render Applicants' method obvious. This is because the combination of Cuttitta and Pandurangi, which teaches "the photoaction of aryl azide compounds to produce singlet nitrenes for phototherapeutic uses" (August 17, 2010 Action), does not result in the tissue damage that thereby treats the target tissue.

For at least these reasons, Applicants respectfully asset that Cuttitta in view of Pandurangi does not render claims 11, 12, and 21-27 obvious, and request the rejection be withdrawn.

CONCLUSION

The application is believed to be in condition for allowance. The fee to Request Continued Examination is simultaneously being paid by electronic funds transfer. If other fees are required, the Office is authorized to charge them to Deposit Account No. 20-0809.

The Examiner is invited to contact Applicants' undersigned representative with questions.

Respectfully submitted,
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